CLINICAL RESEARCH STUDIES

From the Eastern Vascular Society

Prior endovascular abdominal aortic aneurysm repair provides no survival benefits when the aneurysm ruptures

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Objective: **It has been proposed that prior endovascular abdominal aortic aneurysm (AAA) repair (EVAR) confers protective effects in the setting of ruptured AAA (rAAA). This study was conducted to compare outcomes of rAAA repairs in patients with and without prior EVAR.**

Methods: **A retrospective review identified 18 patients with (group 1) and 233 patients without (group 2) antecedent EVAR who presented with rAAA from January 2001 to December 2008. Patient characteristics and perioperative variables were noted and the outcomes were compared. Multiple logistic regression was used to identify factors contributing to morbidity and mortality and Kaplan–Meier analyses to estimate late survival rates.**

Results: **Baseline characteristics were similar between groups. Mean age was 78 years in group 1 and 74.8 years in group 2 ($P = .17$). Men comprised 83.3% of patients in group 1 and 77.3% in group 2 ($P = .77$). Hemodynamic instability at rAAA was noted with similar frequency between groups, 55.6% vs 52.6%, respectively ($P = .99$). Mean time from EVAR to rAAA was 4.0 years and from last follow-up computed tomography (CT) 1.2 years. The devices involved were Ancure (Guidant, Menlo Park, Calif) (9), AneuRx (Medtronic, Minneapolis, Minn) (5), Zenith (Cook Medical Inc, Bloomington, Ind) (3), and Excluder (W.L Gore, Flagstaff, Ariz) (1). Mean preoperative AAA size was 6.4 cm in group 1. All but 1 patient had an endoleak at the time of rupture. Of 14 patients with CT follow-up, only 3 patients had a known increase in size (≥5 mm) and only 3 were known to have an endoleak. Fifteen patients were treated by a single intervention, whereas 3 patients underwent multiple procedures. In group 2, open repair was performed in 218 patients and EVAR in 15. Morbidity (66.7% vs 56.7%) and in-hospital mortality (38.9% vs 36.9%) were nearly identical between groups. One-year survival rates (27.8% vs 48.2%; $P = .15$) were also similar. The mortality rates for EVAR for primary rAAA was 20% as compared to 38.1% for open repair for rAAAs ($P = .27$).**

Conclusion: **rAAA remains a lethal problem in patients with and without prior EVAR alike. An existing endograft provides neither acute nor 1-year survival benefits after rAAA repairs. Prediction of patients at risk for rupture post-EVAR is difficult, as only a minority of patients had a known prior endoleak or sac enlargement. (J Vasc Surg 2010;52:1127-34.)**

Although endovascular aneurysm repair (EVAR) has been proven to be an effective method of treating an abdominal aortic aneurysm (AAA) with superior short-term outcome data by both prospective randomized trials[1,2] and population-based observation studies,[3] it has not completely eliminated AAA rupture in long-term follow-up. Post-EVAR rupture of an AAA occurs from incomplete exclusion of the aneurysm by a variety of endoleaks and endotension. The reported incidence of incomplete exclusion ranges from 6% to 50%.[4] Cumulative annual risk of rupture after EVAR depends on a variety of factors and is estimated to occur between 0.2% and 1.0% per year.[4-7]

Some reports of late aneurysm rupture after EVAR have suggested that a stent graft may confer a survival benefit compared with de novo rupture due to the higher likelihood of hemodynamic stability.[8,9] The reported operative mortality rates for repair of post-EVAR ruptured abdominal aortic aneurysms (rAAA) have ranged, however, from 17% to 62%.[5,9-11] The purpose of this study was to identify any predictive factors of late rupture in patients previously treated by EVAR and to compare

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the outcomes of such an event to patients with a de novo rupture of untreated AAA.

MATERIALS AND METHODS

A retrospective review of medical records at the University of Pittsburgh Medical Center (UPMC) identified 251 consecutive patients who presented with an rAAA without thoracic extension from January 2001 to December 2008. A rupture of the aortic neck into the sac secondary to aggressive balloon dilatation and severe oversizing of the endograft were excluded. Other acute conversions for aorto-enteric fistulae (2 patients) were also excluded. The diagnosis of rupture was made by review of preoperative computed tomography (CT) findings. This was confirmed by a clear description of blood outside the aneurysm wall, either in the retroperitoneum, mesentery, or the peritoneal cavity in those patients undergoing open repair. The initial rupture episode was counted as the index procedure. This study was approved by the Institutional Review Board of the University of Pittsburgh.

Baseline patient characteristics and perioperative variables were recorded. Preoperative shock was defined as systolic blood pressure less than 80 mm Hg or a requirement for preoperative cardiopulmonary resuscitation (CPR). Postoperative renal insufficiency was defined as serum creatinine greater than 2.0 mg/dL.[12] Operative mortality was defined as death within 30 days of operation or in-hospital death, if later than 30 days. Open or endovascular repairs were performed by 14 different vascular surgeons at UPMC during the study period.

Quantitative variables are summarized as mean and SD, and categorical variables as counts and percentages. Baseline characteristics were compared using *t* test for quantitative variables or χ^2 test and Fisher exact test for categorical variables. Univariate analysis was used to assess demographic, clinical, procedural, and postoperative factors associated with operative deaths (Table I). Multiple logistic regression analysis was performed to identify factors that were independently associated with operative mortality. Survival rates were calculated using Kaplan–Meier method, using the log-rank test for comparison of curves, if appropriate. Social Security Death Index was used to supplement data on late mortality. Analyses were performed with the statistical software R, version 2.9.2 under the alpha level of 0.05 (http://www.r-project.org).

RESULTS

The baseline characteristics of the patients are detailed in Table I. A total of 251 patients underwent repair of rAAAs; 18 patients with (group 1) and 233 patients without (group 2) antecedent EVAR. Mean age was similar between the two groups, 78.0 years (range, 59-92) in group 1 and 74.8 years (range, 50-92) in group 2 (P = .17). Men comprised 83.3% of the patients in group 1 and 77.3% in group 2 (P = .77).

Hemodynamic instability, defined as systolic blood pressure <80 mm Hg, at presentation occurred in 55.6% of the patients in group 1 and in 52.6% in group 2 (P > .99). Preoperative CPR was performed with similar frequency, 16.7% in group 1 and 22.2% in group 2 (P = .77). The prevalence of comorbidities was similar between the two groups except the incidence of coronary artery disease and current smoking (Table I).

Table I. Baseline characteristics and demographics comparison of ruptured abdominal aortic aneurysm (rAAA) patients with and without antecedent endovascular aneurysm repair (EVAR)

	Average ± SD (n) or percentage (n)		
Variable	*Group 1 (18)*	*Group 2 (233)*	P
Patient demographics			
Age (years)	78	74.8	.17
Male	83.3	77.3	.77
History of HTN	77.8	79	.99
History of COPD	33.3	32.3	.99
History of CAD	72.2	54.5	.22
History of DM	22.2	15.3	.50
History of PVD	33.3	21.3	.24
History of CVA	5.6	14.5	.48
History of CRF	22.2	14.9	.49
History of HD	0	.5	.99
Current smoking	11.1	34.4	.06
Preoperative characteristics			
Preoperative SBP <80 mm Hg	55.6	52.6	.99
Preoperative CPR	16.7	22.2	.77
Blood chemistry			
Mean hemoglobin (g/dL)	9.9	11.1	.007
Mean platelet	186	213	.27
Intraoperative variables			
RBC transfusion in units	3.26	7.14	<.001
FFP transfusion in units	1.6	4.26	<.001
Platelet transfusion in packs	0.4	1.04	.003
Postoperative complications			
In-hospital death	38.9	36.9	.99
Renal failure requiring HD	26.7	23.9	.76
Myocardial infarction	50	38.3	.43
Tracheostomy	31.2	23.8	.55
Stroke	0	8.8	.62
Intestinal ischemia	18.8	19.8	.99

Data are % (n) or mean ± SD.

CAD, Coronary artery disease; *COPD,* chronic obstructive pulmonary disease; *CPR,* cardiopulmonary resuscitation; *CRF,* chronic renal failure; *CVA,* cerebrovascular accident; *DM,* diabetes mellitus; *FFP,* fresh frozen plasma; *HD,* hemodialysis; *HTN,* hypertension; *PVD,* peripheral vascular disease; *RBC,* red blood cell; *SBP,* systolic blood pressure.

Rupture after a previous EVAR (group 1). Eighteen patients presented with rAAA with the following endoprostheses: Ancure (Guidant, Menlo Park, Calif) (9), AneuRx (Medtronic, Minneapolis, Minn) (5), Zenith (Cook Medical Inc, Bloomington, Ind) (3), and Excluder (W.L. Gore, Flagstaff, Ariz) (1). Indications for original EVAR was for AAA for all except 1 patient (patient #18) who was treated for an isolated right common iliac artery aneurysm with an Excluder iliac limb. Five patients had undergone the initial EVAR at an outlying hospital.

Table II. Detailed description of patients with ruptured abdominal aortic aneurysms (rAAA) with antecedent endovascular aneurysm repair (EVAR), their treatment, and outcomes

Variable	*Post-EVAR Ruptured AAA*
Patients, n	18
Interval from initial EVAR to rAAA	4.0 years
Interval from last follow-up to rAAA	1.2 years
Patients with >2 years since last follow-up to rAAA	3
Patients with rAAA within 1 year of last follow-up	10
Patients with secondary interventions before rAAA	4
Aneurysm characteristics during follow-up	
Mean size at initial EVAR, mm	63.5
Endoleak at last follow-up, (n = 13)	
None	10 (77%)
Type Ia	1 (8%)
Type II	2 (15%)
Unknown (no contrast on CT)	
Sac size at last follow-up (n = 13)	
Increase in sac size	3 (23%)
Decrease in sac size	6 (46%)
No change in sac size	4 (31%)
Aneurysm characteristic at the time of rupture	
Mean size at rupture, mm	67.6
Endoleak at rAAA, (n = 18)	
Type I proximal	9 (50%)
Type I distal	6 (33%)
Type II	2 (10%)
Type III fabric fatigue	2 (10%)
Type III modular disconnection	1 (5%)
Undetermined	1 (5%)

AAA, Abdominal aortic aneurysm; *CT*, computed tomography; *EVAR*, endovascular aneurysm repair; *rAAA*, ruptured abdominal aortic aneurysm.

Post-EVAR rAAA occurred at an average of 4.0 ± 1.9 years (range, ~0.3-7.8) after EVAR (Table II). There were two ruptures within the first year and five within the first 3 years. It should be noted that 1 patient presented with a rupture even after partial endograft explantation. This was a 91-year-old man (patient #1) who underwent an elective EVAR with a Zenith device at age 90. He had type I and type II endoleaks with sac expansion despite two interventions consisting of proximal Palmaz stent (Cordis Endovascular, Warren, NJ) placement and inferior mesenteric artery (IMA) coil embolization, and underwent partial endograft explantation (with the iliac limbs left behind) at 14 months post-EVAR. He recovered from that operation well, but the AAA ruptured 20 months later from distal bilateral type I endoleaks.

All but 1 patient with an rAAA had an endoleak that was confirmed by CT scan or operative findings at the time of the rupture. Isolated type Ia (proximal) endoleaks were noted in 9 patients (of which 2 were associated with migration), type Ib (distal) in 5 patients, and type III in 3 patients. Multiple endoleaks were seen in 3 patients (Ib and II, Ia and III, and Ib, II, and III in 1 patient each). In 1 patient the endoleak was undetermined. Two patients with proximal migration presented with rupture. The first one occurred 10 months after the successful index EVAR with normal baseline CT scan, whereas the second aneurysm ruptured 7.8 years after EVAR. The last CT scan without contrast enhancement obtained 1.7 years before rupture had shown a stable sac size with 10 mm distal migration. This was neither treated nor followed further. Most of the endoleaks at the time of rupture were previously undiagnosed on the last imaging follow-up.

Follow-up CT scans after initial EVAR were available in 14 patients, with contrast enhancement in 11; 3 of the 14 were baseline studies 1 month postoperatively. In 1 patient who had the index EVAR performed at an outlying hospital, the follow-up information was not available. The mean interval from last follow-up imaging to rupture was 1.2 ± 1.1 years (range, ~2 days to 3.9 years). Last follow-up before rupture was within a year in 9 patients, between 1 and 2 years in 5 patients, and more than 2 years in only 3 patients.

Endoleak data were available at last follow-up in 13 patients (Table II). An endoleak had been present in only 5 patients (38%). A type II endoleak was noted in 2 patients, both of which were associated with an enlarging sac, including 1 patient who had an endograft infection. In 1 patient, a type I endoleak was noted with increasing sac size. This patient's aneurysm ruptured 2 days later while he was in the hospital undergoing preparation for further evaluation and treatment. In 3 additional patients, the status of the endoleak could not be ascertained due to the CT scans without contrast enhancement; these were associated with sac growth, shrinkage, and stability in 1 patient each.

Mean preoperative aneurysm size was relatively large, 6.4 ± 1.3 cm in the patients with later rupture after EVAR. AAA sac size analysis was limited to 13 patients who had at least one follow-up imaging beyond the 1-month postoperative study and only 3 patients had a known sac enlargement of more than 5 mm. Sac behavior at last follow-up is detailed in Tables II and III.

Secondary endovascular interventions had been performed in 4 patients before rupture. One patient had undergone partial removal of the stent graft as mentioned above. Two patients had coil embolization of branch vessels and 1 patient required an iliac limb extension.

A total of 23 open/endovascular interventions were performed (Table III). Multiple interventions were undertaken in 3 patients. One patient (patient #1, mentioned above) initially underwent a left iliac limb extension for rupture due to a left type Ib endoleak. However, he continued to bleed requiring multiple units of blood product transfusion over the ensuing 6 days. He then underwent right iliac limb extension. Although he stopped bleeding, he never recovered, and he died in the hospital. One patient (patient #3) underwent bilateral iliac limb extension when he presented with an rAAA. This was unsuccessful and required ligation of the right hypogastric artery via a retroperitoneal incision 4 days later. Despite that, he continued

Table III. Follow-up data on patients with post-endovascular aneurysm repair (*EVAR*) ruptured abdominal aortic aneurysms (*rAAA*)

Pt #	*Gender*	*Age*	*Endograft type*	*Interval from EVAR to rAAA (years)*	*Interval from last F/U to rAAA (years)*
1[a]	M	91	Zenith	2.8[b]	1.7[b]
2	M	77	AneuRx	3.3	3.2
3[a]	M	71	Ancure	2.4	0.4
4[c]	F	74	AneuRx	3.3	1.0
5	F	77	Zenith	0.8	0.7
6	M	73	AneuRx	5.7	1.5
7[c]	F	73	Ancure	0.5	0.1
8	M	85	Ancure tube	4.9	3.9
9	M	82	AneuRx	7.8	1.7
10[c]	M	87	AneuRx	5.8	0.7
11	M	82	Ancure	3.7	2.7
12	M	68	Ancure	4.6	2 days
13	M	59	Ancure	2.6	0.6
14[c]	M	78	Ancure	N/A	N/A
15	M	92	Ancure[e]	5.3	0.4
16[a,f]	M	64	Ancure	6	0.1
17[c]	M	89	Zenith	3.4	0.6
18	M	83	Excluder iliac	4.8	1.3

Pt #	*Cause of rupture*
1[a]	Bilateral type Ib
2	Unknown (ct not done)
3[a]	1: Type Ib, bilateral 2: Type II (from right IIA) 3: Type Ib
4[c]	Proximal collapse with type Ia
5	Type Ia (migration)
6	Type III (left limb disconnection)
7[c]	Type Ia (graft infection with MRSA)
8	Type III (a hole in midbody)
9	Type Ia (migration)
10[c]	Type Ib, left
11	Type Ia (floating device) and type III
12	Type Ib
13	Type Ia
14[d]	Type Ia
15	Type Ib
16[a,f]	1: Type Ib, left 2: Type II 3: Type III
17[c]	Type Ia
18	Type Ia

AAA, Abdominal aortic aneurysm; *CT*, computed tomography; *EIA*, external iliac artery; *embo*, embolization; *EVAR*, endovascular aneurysm repair; *F*, female; *F/U*, follow-up; *IIA*, internal iliac artery; *IMA*, inferior mesenteric artery; *M*, male; *MRSA*, methicillin-resistant staphylococcus aureus; *N/A*, not available; *Op.*, operative; *rAAA*, ruptured abdominal aortic aneurysm.

[a]Underwent multiple interventions.

[b]Endograft was explanted and replaced with a surgical graft at 14 months post-EVAR and presented with rupture 20 months later. The time of endograft explant is considered the last follow-up , although the patient was seen 6 months before rupture without any imaging study. It was considered that there was no endoleak upon open conversion.

[c]Initial EVAR performed at outside of University of Pittsburgh Medical Center system.

[d]Decreased by >5 mm during follow-up but increased back to baseline size on the last follow-up.

[e]Aortouniiliac and cross-femoral bypass.

[f]The patient presented with three separate episodes of rAAA.

Table III. Continued

AAA size @ 1 mo postop (mm)	*AAA size @ last F/U (mm)*	*AAA size change compared to baseline @ last F/U*	*Endoleak @ last F/U*	*No. of prior intervention(s) and details*	*Details of treatment*	*Outcome*
89	N/A[b]	N/A[b]	No[b]	1: Palmaz stent for type Ia leak 2: IMA coil embo; coil embo for recurrent type Ia leak 3: Explantation (main body only)	1: Left iliac limb extension 2: Right iliac limb extension Explantation	Op. death Died @ 4 mos
51	N/A	N/A	No	None	1: Bilateral iliac Wallgraft placement 2: Ligation of right IIA 3: Interposition grafting of the left limb; ligation of a lumbar artery	Died @ 7 mos
57	62	Increased	Type II	1: Lumbar artery coil embo	Multiple proximal extender cuff (4, Zenith and Excluder) and Palmaz stent placement	Op. death
70	46	Decreased	No	None	Proximal extension cuff (Zenith) with Palmaz stent	Died @ 8 mos
61	N/A	N/A	No	None	Left limb extension	Alive @ 19 mos
77	80	No change	No	1: Right limb extension @ 2 years	Explanted (a portion of right limb left behind)	Died @ 5 mos
45	50	Increased	Type II	None	Explanted	Op. death
53	44	Decreased	No	None	Aortic extender: Zenith cuff and 3 Excluder cuffs	Op. death
60	59	No change	No contrast	None	Left iliac limb extension	Died @ 14 mos
60	44	Decreased	No contrast	None	Explanted	Alive @ 72 mos
72	N/A	N/A	No	None	Right iliac limb extension with a Wallgraft	Alive @ 86 mos
50	37	No change[d]	No	None	Explanted (bilateral limbs left behind)	Alive @ 45 mos
94	68	Decreased	No	None	Explanted (right limb left behind)	Died @ 3 mos
N/A	N/A	N/A	N/A	Unknown	Extended with AneuRx limb and Wallgraft	Op. death
55	45	Decreased	No	None	1: Left iliac limb extension; coil embolization of the distal landing zone 2: Open ligation of lumbar artery 3: Partial explantation leaving the proximal and iliac limbs behind; bifurcated graft sewn end-side to bilateral EIA with proximal iliac limbs ligation	Op. death
65	63	No change	No	1: IMA and right lumbar coil embo	Endovascular repair attempted but patient expired on table	Op. death
59	65	Increased	No contrast	None	EVAR with an Excluder bifurcated graft	Died @ 1.5 mos
62 (iliac); 36 (aorta)	62 (iliac); 43 (aorta)	Decreased iliac; Increased aorta	No	None		

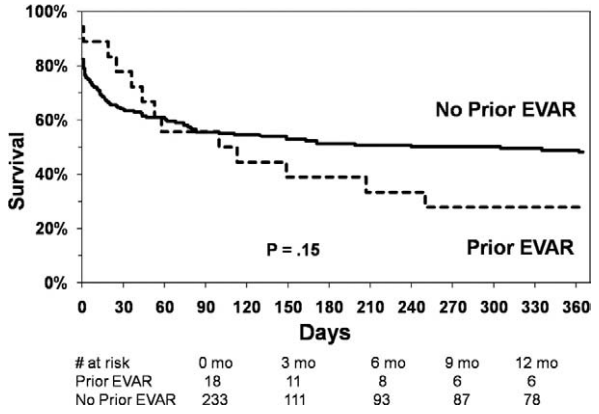

Fig. Kaplan–Meier estimate of probability of survival in patients who presented with ruptured abdominal aortic aneurysm (rAAA) with and without antecedent endovascular aneurysm repair (*EVAR*).

to bleed and underwent open conversion with left iliac limb interposition grafting and ligation of a bleeding lumbar artery. He was discharged to a nursing home and died 4 months later. The last patient (patient #16) underwent endovascular left iliac limb extension and coil embolization of the distal landing zone when he first presented with an rAAA. He was discharged to home the next day. Three days later, he presented to the emergency department with another episode of severe abdominal pain. He was explored for persistent bleeding and found to have an rAAA with fresh thrombus in the retroperitoneum. The aneurysm sac was opened and a type II endoleak from a lumbar artery was noted; no evidence of a type I endoleak was present. The artery was ligated and the sac closed without removal of the endograft. The patient did well and was discharged to home 13 days later only to return 5 days afterward with yet another rupture. The etiology was unclear, but he was noted to have diffuse bleeding through the fabric of an Ancure device. The graft was explanted except for the segments at the proximal and distal sealing zones and replaced with a surgical graft. He developed other complications and died in the hospital.

The remaining 15 patients underwent a single therapeutic intervention. Open conversion was performed in 7 patients (complete explantation in 2 patients and partial in 5) with a surgical graft placement, whereas endovascular intervention was performed/attempted in 10 patients (proximal aortic extension in 3 patients, iliac extension in 5, EVAR with a bifurcated endograft in 1, and a failed attempt in 1 patient who died on the operating table).

Rupture without a previous EVAR (group 2). Of 233 patients in group 2, 218 patients underwent open repairs, whereas the remaining 15 patients were treated by EVAR. Overall, the in-hospital mortality rate was 36.9%. Patients treated with EVAR seemed to have a slightly lower mortality rate, 20.0%, than those who underwent open repair, 38.1% ($P = .27$).

Comparison of group 1 and 2. No significant differences in perioperative complication rates (66.7% vs 56.7%; $P = .47$) and operative mortality rates (38.9% vs 36.9%; $P = .99$) were noted between groups 1 and 2, respectively. Only intraoperative blood product requirements were significantly lower in group 1 compared to group 2 (Table I).

The probability of survival at 1 year was also similar between groups, 27.8% (group 1) vs 48.2% (group 2; $P = .15$; Fig). The multivariate analysis of operative mortality with the multiple logistic regression model revealed that advanced age and preoperative CPR were independent predictors of death ($P < .001$).

DISCUSSION

This study represents the largest single-center experience of post-EVAR rAAA and shows that the operative mortality rate for rAAA with antecedent EVAR is not necessarily lower than that of de novo rAAA. Most patients do not have a known endoleak or sac enlargement, rendering prediction of late rupture after EVAR difficult. Rupture remains a potential, albeit rare, catastrophic event even in patients with sac shrinkage and no endoleaks.

The reported operative mortality rates for post-EVAR rAAA range widely from 16% to 67%.[5,9-11,13-15] May et al[13] reported a significantly reduced mortality rate of 17% with open repair of post-EVAR rAAA compared with 54% in patients with de novo rAAA, and attributed its survival benefit to the reduced blood loss and relative hemodynamic stability. Several authors have postulated that an existing stent graft improves the chance of survival by providing hemodynamic stability.[8,9,13] Coppi et al,[9] in their comparative analysis of 14 post-EVAR rAAA and 155 de novo rAAA, noted less frequent hemodynamic instability (the only predictor of death identified in their series) in patients with antecedent EVAR, but similar overall 30-day mortality rates between the two groups (28.5% vs 38.7%, respectively). Therapeutic modalities (open vs endovascular) did not have any impact on death rates between the two groups either. Nonetheless, they supported the theoretical survival advantage in the presence of an endograft. However, our experience does not support this assumption and is more in line with a worldwide literature review recently published by Schlösser et al.[5] In a compilation of 270 late AAA ruptures after EVAR, they reported a 43% (69 of 164) operative mortality rate. Fransen et al[10] also reported a 62% operative mortality rate in a survey of 34 rAAAs after EVAR from 4291 patients in the EUROSTAR registry. In the present series, even the incidence of hemodynamic instability did not differ between the two groups, although blood product transfusion requirements were significantly lower in patients with rAAAs after EVAR. The nearly identical operative mortality rates between the two groups suggest that the late ruptures after EVAR is as lethal as primary rAAA.

Predictors of late AAA rupture after EVAR in the literature have been found to include: large initial AAA diameter,[7,16-18] type I and III endoleaks,[6,7,13,19-23] graft migration,[6,10,24] sac growth,[7] and poor compliance with

follow-up.[25] Of these, endoleak is the most common culprit for late ruptures.[5,9,10] In the present study, all post-EVAR ruptures had an endoleak, but unfortunately, most of them were not there at last follow-up. Timely detection and treatment of type I or type III endoleaks and type II endoleaks associated with sac growth should be a standard practice. Type II endoleak is not necessarily a benign finding,[20] as it may develop and resolve spontaneously. In the AneuRx trial, 5 of 7 patients with post-EVAR ruptures showed no evidence of endoleak at last follow-up before ruptures.[18] Similarly, 77% of our patients showed no evidence of endoleak at last follow-up in this study. It should be noted, however, that follow-up imaging was available for review in only 13 of 18 patients (72%) in the present study, although 9 of them were within 12 months of rupture. The majority of endoleaks found at the time of rAAA were new. Others have also noted that up to 40% of patients had no abnormalities detected at last follow-up before rupture.[5,10] The absence of endoleak during follow-up does not mean that all is well.

Another parameter that is used and accepted as a surrogate marker for a successful EVAR is AAA sac shrinkage. Its value as a reliable sign of long-term success is debatable. Shrinking AAA sac may re-expand over time.[26] Reports from the EUROSTAR registry and literature review indicate that about 60% of patients showed either shrinking or unchanged sac size at last follow-up.[5,10] The findings in the present study are in agreement with these reports; of 13 patients in whom size comparison was possible, only 3 patients had shown sac growth at last follow-up before rupture. A shrinking or stable aneurysm sac size does not necessarily reflect cure of the aneurysm and should not result in a cessation of follow-up.[9,25]

Although sac size change and endoleak were not uniformly present in patients who ultimately rupture after EVAR, a large initial size of the AAA treated is fairly prevalent among these patients. Not only is it the primary determinant of the risk of primary aneurysm rupture, but also seems to be a strong predictor of comorbidities,[7,18] increased operative risk,[7,27,28] and post-EVAR outcome.[5,7,9,13,15,18] After EVAR, large AAA is associated with late ruptures, type I endoleak, and aneurysm-related death. Zarins et al,[18] in a review of 923 patients treated with AneuRx stent grafts, showed that large preoperative AAAs (≥6.0 cm) was the only independent predictor of the aneurysm-specific endpoint of rupture, AAA-related death, and conversion; the probability of freedom from rupture at 5 years was 100% for AAAs <5 cm, 97% for AAAs between 5 and 6 cm, and 93% for AAAs >6 cm. Large AAA was also more likely to enlarge post-EVAR.[18] Peppelenbosch et al,[7] in a survey of data from 4392 patients in the EUROSTAR registry, showed that the ratio of aneurysm-related to unrelated death was about 50% in patients with large (≥6.5 cm) preoperative AAA diameter as compared with 28% and 23% in the smaller aneurysm groups. The mean preoperative AAA size of those that ruptured after EVAR is 6.5 cm in the Lifeline Registry and AneuRx study, whereas the mean diameter of aneurysms treated was 5.6 and 5.7 cm, respectively.[18] In the present study, the mean AAA size that ruptured after EVAR was 6.4 cm, resonating the findings of others.[7,10,13]

Continued follow-up after EVAR is critical regardless of the apparent success of the operation. As can be seen from this series, the events and changes that lead to rupture can be catastrophic and happen rapidly between follow-up periods, and there were no tell tale signs that could portend a future rupture. As such, it is not easy to provide a recommendation regarding a follow-up protocol. In fact, surveillance protocol after EVAR is evolving. The optimal schedule that may minimize rupture is not well defined, and unfortunately, this report indicates that rupture can happen even after a recent normal follow-up scan. At present, our protocol includes a baseline CT scan at 1 month. If there is no evidence of endoleak or device-related abnormalities, a follow-up CT scan is obtained at 1 year. A stable or shrinking aneurysm then usually prompts a switch to Duplex scan as the modality of follow-up.

There are several limitations of this study including the limitations of retrospective data collection, incomplete follow-up, heterogeneity of pathophysiology, and treatment modalities in a small sample size of post-EVAR ruptures, which limited statistical analysis. Many devices represented in this series are either no longer available or in diminishing use. The findings may not be applicable to newer generation devices as technology continues to improve.

CONCLUSIONS

Ruptured AAAs with antecedent EVAR are as lethal as de novo types. The endograft does not seem to provide any survival benefits when the aneurysm ruptures. Prediction of patients at risk for post-EVAR rupture is difficult, as it is often a new catastrophic failure that may occur despite a previous appearance of a successful exclusion of the sac.

AUTHOR CONTRIBUTIONS

Conception and design: JC
Analysis and interpretation: JC, TP
Data collection: JC, JK
Writing the article: JC
Critical revision of the article: JC, MM, RR, RC
Final approval of the article: JC
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